

55th Annual Meeting of the American Academy of Neurology

29 March – 5 April, 2003; Honolulu, Hawaii

The 55th Annual Meeting of the AAN coincided with the International Spam Festival (Spam – spiced ham). 7200 delegates registered but the Iraqi war and fear of SARS took their toll and well under that number actually attended.

The scientific programme, as usual was held mainly on Tuesday, Wednesday and Thursday while the bulk of the education programme took place on Sunday, Monday and Friday. There was an extensive choice of nearly 200 teaching sessions ranging from breakfast and after dinner seminars to all day sessions. It seemed to me that the teaching programme was virtually identical to the previous meeting I attended in Denver. The scientific programme comprised 1300 platform or poster presentations out of a total of 2600 submitted. It was a bit disappointing that about one in five posters was withdrawn. However, there was no shortage of interesting material presented.

The Oxford group presented data on the clinical characteristics of ten myasthenic patients with MuSK (muscle specific tyrosine kinase) antibodies. Anti-MuSK antibodies are seen in about 40% of patients with seronegative generalised myasthenia gravis (but not in pure ocular myasthenia). These patients were all female and they tended to have weakness of neck (particular neck extension), shoulder and respiratory muscles with little ocular involvement. The response to pyridostigmine was variable while thymectomy and azathiopine were ineffective. All patients however responded well to plasma exchange but no comment was made about response to ivIg. This important paper therefore gave useful information about the clinical characteristics of these patients and also their different responses to treatment.

The Parkinson's Disease study group reported further on the Elldopa trial. This was set up to see if L-Dopa influences the rate of progression of Parkinson's Disease. Three hundred and sixty one patients were enrolled with early Parkinson's Disease who did not require symptomatic therapy. They were allocated in a double blind fashion, to either placebo or three different dosing regimes of L-Dopa. Regular assessments were made during the study and after forty weeks, treatment was withdrawn with the final assessment two weeks later. Patients treated with L-Dopa showed a dose dependent clinical effect based on the UPDRS scores and this persisted two weeks after treatment was withdrawn. Neuro-imaging was carried out in 142 patients with the striatal dopamine transporter

being assessed by Beta CIT uptake measured by SPECT. The percentage decline of Beta CIT uptake in the striatum was significantly more pronounced in the L-Dopa groups than the placebo group. These rather contradictory findings clearly need further investigation; there remains continuing concern about the harmful effects of long term L-Dopa treatment.

There were not surprisingly a number of papers on West Nile virus infection which has been gradually spreading across the USA over the last few years. This is a mosquito born viral infection which usually causes a sub-clinical illness. However there is a wide spectrum of neurological presentations which include meningoencephalitis with or without focal deficits, lumbosacral plexopathy, Guillain-Barre like illness and a cerebellar syndrome. I am unaware of any cases in the UK but we need to bear this infection in mind in patients with acute neurological problems who have recently been abroad.

My own area of interest is in epilepsy and there were some interesting papers. One study followed up twelve patients with refractory epilepsy and evidence of mesial temporal sclerosis who declined surgery. These patients had repeat MRI scans with volume measurements after an interval of 2.5-5.2 years. Three patients became seizure free while nine remained intractable. A significant decline in ipsilateral hippocampal volume occurred only in patients who had continuing seizures while there was no change on the contralateral side. This would suggest that continuing seizures cause progressive hippocampal atrophy. Perhaps we should give more thought to repeating MRI scans on patients with continuing seizures who have focal EEG changes and either normal or minimally abnormal MRI scans.

I am not a golfer myself but I was interested in a study of six professional and seven amateur female golfers who were asked to mentally execute their pre-shot routine to a target pin and then to imagine their appropriate swing. They underwent functional MRI during these mental activities and some interesting differences emerged. The volume of brain activated in professional golfers was smaller in all active regions and amateur golfers activated a number of additional areas. These included the limbic system and amygdaloid complex, basal forebrain and basal ganglia. The authors concluded that the neural networks in professional golfers was more focused suggesting a more specialist and efficient network whereas the amateur golfers activated other areas involved in learning and emotional expression. I will pass this vital information to my golfing neurological colleagues although I suspect they will say (as they are all male) that this has something to do with the peculiarities of the female golfing brain.

Honolulu itself is a pleasant uncommercialised city with a fascinating array of flora which include a variety of palm trees, the delicate Monkey Pod trees, Hibiscus (the state flower of Hawaii), bougainvillea, ginger plants and various ferns. I did not of course have time to go on a submarine excursion nor a helicopter tour as I was too busy extracting pearls of information from the conference. My surfing instructor however did tell me that I would need at least another fourteen days to be able to 'do it standing up;' I have my doubts. The trouble is that I will be 105 when the next AAN meeting is held in Hawaii.



Venue: Hawaii Convention Centre



Honolulu sunset

Picture: Chuck Painter

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